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THE LYMPHATIC DRAINAGE OF THE PARANASAL SINUSES.*†

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INTRODUCTION.

The lymphatics have often been suggested as the probable pathway along which infection extends from the paranasal sinuses to the lungs, a recent writer even stating that "lymphatic drainage from an infected sinus causes the tracheal cough so characteristic of sinus infection." A possible relation between chronic sinusitis and bronchiectasis is not to be doubted, but the question of the mechanism by which infection spreads from the sinus to the lung has not been answered.

Our clinical and anatomical experience has led us to conclude that the respiratory passages are the probable pathway for sinolung infection, aspirated bacteria being carried down the pharynx in the postnasal discharge. To be sure, other pathways for the spread of infection from sinus to lung have been postulated: *1.* The purely lymphatic route — drainage by the lymphatics to the pharynx, with subsequent retrograde extension along the lymphatics draining the lungs; *2.* the combined lymphatic-vascular route — drainage by the pharygeal lymphatics and great veins to the right side of the heart, then by the pulmonary arterial circulation to the lungs; *3.* direct extension of the infection by lymphatics and poten-

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tial tissue spaces. This paper will present evidence that these alternate pathways for the spread of infection are improbable.

The second alternate route just mentioned — the combined lymphatic and vascular pathway — has been emphasized particularly by Mullin[1] and by Larsell and Fenton.[2] Mullin investigated the lymphatic drainage of the maxillary and frontal sinuses in rabbits, and Larsell and Fenton the lymphatic drainage of the frontal sinus and of the pharynx in cats.

Mullin's rabbits were anesthetized with ether, and a hypodermic needle was pushed through the skin, the fascia and the thin external wall of the superior maxillary bone, until the point of the needle was presumably in the cavity of the maxillary sinus. Relatively large quantities (0.5 cc. to 2 cc.) of India ink were then injected into the sinus cavity. He found that some ink always escaped into the nasal cavity and ran out from the nostril, and that "there was also some staining of the soft tissues of the face outside the sinus." He probed the nose with an applicator, but it was, of course, impossible to swab away all of the excess ink which spilled over into the nasal cavity. Furthermore, since the rabbits were under ether anesthesia, undoubtedly some ink dripped into the nasopharynx, and it is not surprising that some of it was found in the lungs. Mullin reported that the internal jugular lymph nodes, the submaxillary node, and the bronchial nodes were all deeply stained with India ink. Since the submaxillary nodes lie in the pathway of the lymphatic drainage of the face and the anterior part of the nasal cavity (Poirier and Cuneo[3]), and since, by Mullin's technique, the soft tissues of the face were stained with ink, it was to be expected that the submaxillary nodes would contain ink. The present paper will attempt to show that the submaxillary nodes do not drain the paranasal sinuses. Mullin concluded that India ink injected into the sinuses is drained by the lymphatic system and then is carried by the blood stream to the lungs.

Larsell and Fenton anesthetized cats with either pentobarbital or dial in order to prevent the large amount of mucous secretion in the air passages which results from the use of ether in cats. One type of experiment consisted of

exposing the bony wall above the eye, chipping away the bone to expose the mucous membrane of the frontal sinus and, with bone wax, fixing a needle in position between the membrane and the bone. A solution of trypan blue was allowed to drain into the membrane at 50 mm. water pressure. Another method was to inject a suspension of Prussian blue or a solution of potassium ferrocyanide into the posterior wall of the pharynx. In these experiments apparently, relatively large amounts of injection medium were introduced. In some of the experiments, in which the colloid material was injected into the posterior wall of the pharynx, retropharyngeal tissue spaces were filled and dissected down to the thorax. Larsell and Fenton concluded that colloid material may reach the lungs by the combined lymphatic and blood-vascular route.

Our knowledge of the lymphatic drainage of the paranasal sinuses in the human rests largely upon the investigations of Poirier and Cuneo,[3] Most[4], Marc André[5] and Grünwald.[6] In these studies the lymphatics of the nasal cavity were injected in the fresh cadaver mainly by the method of Gerota, which utilizes a turpentine and ether suspension of Prussian blue. The reports agree that the submucosal lymphatics of the paranasal sinuses converge toward the ostia, then drain into the lymphatics of the mucous membrane of the lateral wall of the nasal cavity, and eventually into the parapharyngeal, retropharyngeal and deep cervical lymph nodes. The present report submits evidence in general agreement with this description, adding some details on the disposition of the lymphatics in the lateral wall of the nasal cavity and nasopharynx.

METHODS.

Because we felt that the evidence presented by the experiments of Mullin and of Larsell and Fenton was inconclusive, we investigated the question of the lymphatic drainage of the paranasal sinuses, both experimentally in the living rabbit and by injection methods in unembalmed newborn and adult human cadavers. The rabbits were anesthetized with nembutal, and the maxillary sinuses opened wide to secure good exposure. The mucous membrane of the maxillary sinus in the rabbit is extremely thin — thinner, in fact, than a

No. 27 needle; injection of more than a fraction of a minim dissects the mucous membrane away from the bone and causes extensive bleeding. Minute quantities, therefore, of Higgins India ink, either full strength or diluted with physiological saline solution, were injected under the mucous membrane. We used a No. 27 hypodermic needle and a 1 cc.

Fig. 1. Lymphatics demonstrated by injection of India ink into the mucous membrane of the middle turbinate. These vessels drain into the parapharyngeal lymph nodes, piercing the pharyngeal wall obliquely beyond the apparent point of their termination.

tuberculin syringe graduated in hundredths, usually injecting not over 0.03 cc. at any one point.

The cavity of the sinus was then carefuly swabbed to make sure that no free ink remained in the sinus; free ink would be carried into the nasal cavity with the flow of mucous secretion. The incision was then closed and the rabbits were allowed to recover, and were killed one day to four weeks after the injection. They were autopsied under the dissecting

microscope, and the lymph nodes and lungs were studied histologically. Fourteen rabbits were used in this study.

For the injection studies in the human, the heads of the cadavers were divided sagittally, near the midplane, with a band saw. We attempted to inject the lymphatics of the nasal and pharyngeal mucosa with diluted India ink, inserting a

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Fig. 2. Lymphatics demonstrated by injection of India ink into the mucous membrane of the inferior turbinate. These vessels also drain the mucous membrane of the middle meatus. These vessels drain into the upper deep cervical (jugular) lymph nodes, below the point of apparent termination.

No. 27 needle directly underneath the mucous membrane. The injected lymphatics were followed to the parapharyngeal, retropharyngeal, and jugular lymph nodes. In an endeavor to fill the efferent lymphatics, these nodes were injected by stick injection. The lymph node stick-injection was also tried in living and in recently killed rabbits. Eight adult human heads and 10 still born baby heads were studied by injection.

All injections were studied with the binocular dissecting microscope at magnifications of 9 to 27 diameters.

OBSERVATIONS.

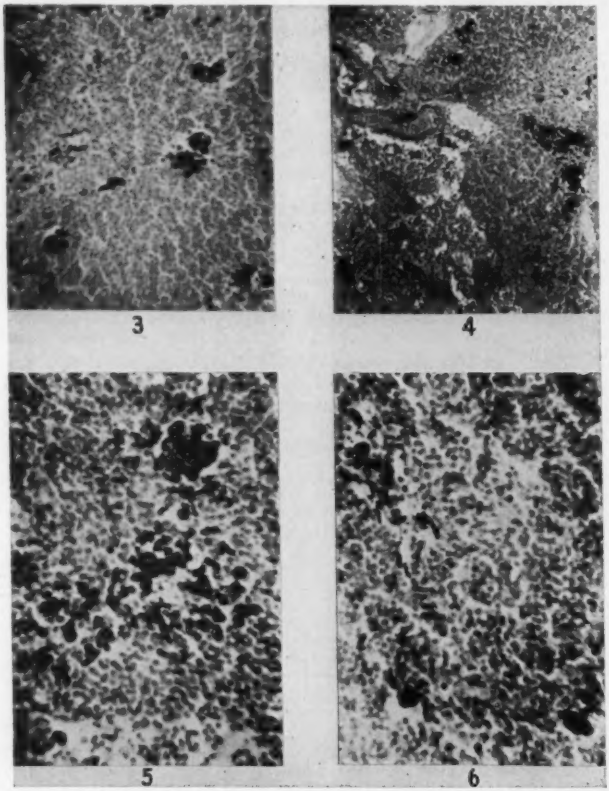
In rabbits in which very minute quantities of ink (0.1 cc. or less) were injected into the mucous membrane of the maxillary sinus, ink was found only in the deep cervical or internal jugular nodes. This node in the rabbit corresponds to the entire chain of upper deep cervical lymph nodes in man, and is usually single, one on each side, about 1 cm. long and 3 to 4 mm. in diameter. We never found any ink in the lower deep cervical or the pulmonary lymph nodes, or in the lungs, even in animals in which larger amounts of ink had been injected.

In two rabbits in which an attempt to inject more than 0.2 cc. of ink into the antral mucosa had resulted in some staining of the soft tissues of the face, the submaxillary lymph nodes contained ink. These nodes, however, never contained ink if the injection was carefully restricted to the maxillary sinus. In these two rabbits, and in a third which received a total of 0.2 cc. of ink, the mucous membrane of the lateral nasal wall, around the ostium of the maxillary sinus, and in the direction of the Eustachian tube, and of the lateral wall of the nasopharynx, was discolored by ink. In one of these rabbits, in which the mucous membrane of an ethmoid cell had also been injected, two lymph nodes back of the pharynx, 2 to 3 mm. in diameter, about 1 cm. superior to the jugular node, and both jugular nodes, were stained.

In all of the animals in which sufficient India ink reached the jugular node, the jugular node of the opposite side also contained some ink; however, the drainage from the sinuses appeared to be rather slow, very little ink having reached these nodes in less than a week's time. Occasionally, in animals receiving sufficient injection, the lymphatic plexus in the lateral nasal or pharyngeal wall contained ink. Several days after the injection, ink was still found in these lymphatics, while the jugular nodes contained no ink until after this time. We saturated a wad of cotton with full-strength India ink, and allowed the wad to remain in the sinus of one animal for a week. Very little of the ink was absorbed, the internal jugular nodes containing almost none.

The protocols of three typical experiments follow:

Rabbit No. 3: Under nembutal anesthesia the right maxillary sinus of a 3-kilo rabbit was exposed. The antral mucosa was injected in five places with a total of 0.2 cc. of whole India ink. Some ink escaped from the



Figs. 3 and 4. Sections of jugular lymph nodes six days after injection of 0.1 cc. India ink under the mucous membrane of the maxillary sinus in the rabbit.

Fig. 3. Lymph node of the same side as injected maxillary sinus.

Fig. 4. Lymph node of the opposite side.

Figs. 5 and 6. Sections of jugular lymph nodes two weeks after the injection of 0.2 cc. undiluted India ink under the mucous membrane of the maxillary sinus in the rabbit.

Fig. 5. Lymph node of the same side as injected maxillary sinus.

Fig. 6. Lymph node of the opposite side.

puncture points and was immediately wiped away, so that no free ink was left in the antral cavity. The rabbit was killed 72 hours later. At autopsy, the mucosa of the right antrum was found to be much discolored by India ink. There was no free ink in the lumen. The rabbit's head was

then sawed in half, disclosing the lateral wall of the right nasal cavity. Immediately above the maxillary ostium and between the ostium and the Eustachian tube, the mucosa was stained with India ink. Under the dissecting microscope, some of this ink was observed to be underneath the periosteum. There was no other discoloration of the mucous membrane in the nose or throat. The right ethmoid sinuses and the right frontal sinus, and all of the sinuses on the left side, were exposed and found to be normal in appearance. The tonsils contained no India ink grossly or microscopically. The deep cervical lymph nodes of both sides were found to be heavily stained with India ink. No ink was found either on gross or microscopic examination in the lower part of the neck or in the chest; the lungs contained no ink.

Rabbit No. 5: Using the same approach as described above, we injected the left maxillary sinus and an ethmoid sinus of a 2.5-kilo rabbit with 0.4 cc. of India ink, much of which spilled into the antral cavity. In hastily swabbing the free ink, we stained some of the soft tissues of the face around the operative wound. The rabbit was killed and autopsied one week later.

Neck: Both superficial submaxillary nodes and both deep cervical nodes were deeply stained with India ink. The lymphatic channels in the neck lying alongside the internal jugular vein and two inferior superficial cervical lymph nodes, one on each side, were stained. Two very small retropharyngeal nodes close to the midline were deeply stained with India ink.

Nose: No ink was found in the sinuses on the opposite side. On the same side (left), the mucous membrane of the maxillary sinus and the ethmoid sinuses was deeply stained. The frontal sinus contained no ink. The floor and entire septal wall of the left nasal cavity contained ink.

Nasopharynx: The left lateral wall showed a dense lymphatic plexus deeply stained with ink. This plexus disappeared abruptly in the lower part of the nasopharynx. There was no ink in the rest of the pharynx or in the larynx. The lungs contained no ink.

Rabbit No. 7: The mucosa of the right maxillary antrum was injected at three points with a total of 0.1 cc. undiluted India ink. The rabbit was killed and autopsied two weeks later. The antral mucosa still contained India ink, but no ink was seen under the low power dissecting microscope in the mucosa of the lateral wall of the nasal cavity or nasopharynx. Both jugular lymph nodes contained ink. No ink was found on histological examination in any of the tissues of the lower part of the neck or in the lungs.

In most of the cadaver experiments, the blood capillaries and the veins filled with India ink before the lymphatics were injected; however, in a few cases we succeeded in demonstrating the lymphatics draining the mucosa of the middle and inferior turbinates. The middle turbinate and superior meatus are drained by two to four large lymphatic vessels which run superior to the ostium of the Eustachian tube and penetrate the lateral wall of the nasopharynx, to empty into the parapharyngeal and lateral retropharyngeal lymph nodes.

The inferior turbinate and the middle meatus (and *pari passu*, the paranasal sinuses) are, to a large extent, drained

by four or five vessels which run anterior and inferior to the opening of the Eustachian tube and obliquely downwards through the wall of the pharynx. They then end in the internal jugular nodes, primarily one large one at the level of the bifurcation of the common carotid, just inferior to the insertion of the posterior belly of the digastric muscle into its central tendon. There is some anastomosis of lymphatics in the space between the choanae and the tubal orifice.

The oblique course of the lymphatics through the lateral wall of the nasopharynx to reach the parapharyngeal and deep cervical lymph nodes explains the apparent abrupt disappearance of the lymphatic plexus beneath the mucosa. This apparent termination of the lymphatics was noted also in the rabbit (rabbit No. 5).

Within the maxillary sinus, we have thus far succeeded in injecting only the veins. These converge toward the ostium, and drain into the mucosal veins of the lateral nasal wall. Since submucosal lymphatic plexuses in general parallel the course of neighboring venous plexuses, we are inclined to accept the following description of Rouvière[7] (page 69).

"The lymphatics of the maxillary sinus anastomose with each other, converging beneath the mucosa towards the opening of the maxillary sinus and towards the membranes which obliterate the accessory openings of the sinus. In this manner the vessels arrive in the middle meatus where they unite with the lymphatics of that region; the latter run toward the pharyngeal opening of the Eustachian tube."

Our description of the lymphatics in the lateral wall of the nasal cavity likewise agrees essentially with that of Rouvière[7] (page 68), except we have demonstrated some upper lymphatics draining into the parapharyngeal lymph nodes.

"The collecting trunks from the respiratory region of the nasal fossae are directed backward and outward beneath the superior mucosa of the soft palate towards the pharyngeal orifice of the Eustachian tube. En route, these vessels receive the lymphatics from the superior surface of the soft palate. Moreover, the most medial collecting trunks anastomose in the median line behind the septum with their fellows of the opposite side. The collecting trunks thus assembled in front of the pharyngeal orifice of the Eustachian tube, by their anastomoses at this point, most often, form a small lymphatic plexus."

The stick-injections of the deep cervical nodes demonstrated the lower cervical lymphatics which empty into the jugular trunks. These lie alongside the internal jugular vein. None of the lymphatic vessels could be injected in a retrograde direction.

DISCUSSION.

Our experiments with rabbits indicate that the lymphatic drainage of the maxillary sinus is a fairly slow process, and is confined to the lymphatics of the lateral nasal and nasopharyngeal walls, vessels which empty into the retropharyngeal and jugular nodes; and further, that, under ordinary conditions, all colloidal material is trapped in the nodes and does not reach the blood stream. Small doses of India ink, therefore, having been used, no ink was found in the lungs or bronchial lymph nodes. The submaxillary nodes contained ink only when tissues of the face were injected with ink.

It is our opinion that when injected material was found in the lungs, as reported by Mullin and by Larsell and Fenton, its presence was due entirely to the huge doses of injection material used by these investigators. In addition, in Mullin's animals, some of the ink undoubtedly reached the lungs by aspiration of postnasal drippings. Larsell and Fenton cannulated the tracheas of some of their animals to exclude the tracheal route, but they flooded and overwhelmed the lymphatic system with amounts of injection mass out of all reasonable proportion to the size of the animals. When smaller amounts of injection material are used, the foreign material is trapped by the lymph nodes. Under ordinary conditions, either physiological or pathological, the flow of lymph is relatively slow, and it is improbable that an infection would extend by the combined lymphatic-blood vascular route.

The clinical lesson which we learn from these experiments is that the lymphatics play no part in the relationship between infections in the sinuses and lungs. We feel that dependent drainage from the upper respiratory tract with its mechanical obstruction and irritation is the deciding factor. The persistent explosive coughing effort to remove this accumulation so injures the bronchi that they do not return to normal.

It is also our belief that there is a certain definite type of individual who is predisposed to this misfortune. In our experience, these pathologic changes may be prevented by appropriate treatment. There is often in these patients a definite allergic family history. In early childhood there is a tendency to repeated upper respiratory infections, from which there is a delayed recovery. The nose is blocked and upon its

floor is a thick tenacious grayish "white-of-egg" secretion. Forcible blowing of the nose does not dislodge it. If this secretion can be removed at frequent intervals and the dependent drainage forestalled, the damage can be prevented.

In practice the nose is carefully shrunken and cleaned at home in the morning, and with especial care at night. During an attack the child is brought daily to the office where the nose is sprayed with ephedrine and weak cocaine solution. After the nose is shrunken, a fine-tipped metal aspirator is used to remove all secretion, special attention being given to the floor of the nose. Children 3 to 4 years of age do not object to this treatment. A cylindrical dilatation of the bronchi was observed on some. These were hospitalized and bronchoscopically aspirated. At present, from Roentgen studies, these lungs appear normal. The longest any of these patients have been under observation is 10 years. If the dependent drainage is controlled, we believe a good, though guarded, prognosis can be given.

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EFFECTS OF CERTAIN DRUGS UPON CILIARY ACTIVITY OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY TRACT.*

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Considerable experimental work has been done during the last 10 years to show the effects of certain drugs, commonly used in office practice, upon the cilia of the mucous membrane of the upper respiratory tract.

Studies have been made upon the mucous membrane of living animals and upon extirpated human mucosa. Since results obtained in these instances were practically the same, we have in this study used only extirpated tissue from the mucous membrane of the nose.

The drugs used were 0.5 per cent solution of chlorbutanol in normal saline; saturated solution of sulfanilamide (0.9 per cent); 5 per cent isotonic solution of sodium sulfathiazole (freshly prepared); 5 per cent solution of sodium sulfathiazole without sodium chloride to make it isotonic; 2.5 per cent sulfadiazine in triethanolamine and butoben; triethanolamine and butoben (solvent only), sulfanilamide powder, sulfathiazole powder, sulfadiazine powder, and penicillin, 5,000 units per cubic centimeter of normal saline.

In addition, the effects upon the cilia of two new vasoconstrictors, 2-aminoheptane sulfate (Tuamine sulfate) and 2 amino-4 methyl hexane sulfate (Fouramine sulfate) were observed and compared with ephedrine sulfate.

For some years we have used freshly made isotonic solutions of ephedrine sulfate, since it was known that the preservative, chlorbutanol, stopped ciliary action after immersion. Experiments in which a 0.5 per cent solution of chlorbutanol in normal saline was used upon extirpated tissue revealed instant stopping of cilia in the 11 cases studied, but in all instances action was resumed in from two to five minutes after washing in Locke's solution. Time of immer-

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sion varied from four to 60 minutes. The cilia responded sluggishly but eventually completely recovered even when immersed for long periods.

TABLE 1. DATA ON 0.5 PER CENT SOLUTION OF CHLORBUTANOL IN NORMAL SALINE.

Time Cilia Stopped After Immersion	Time of Immersion	Recovery Time After Washing in Locke's Solution
Instant	5 minutes	3 minutes
Instant	20 minutes	3 minutes
Instant	20 minutes	2 minutes
Instant	17 minutes	3 minutes
Instant	15 minutes	5 minutes
Instant	60 minutes	5 minutes
Instant	60 minutes	5 minutes
Instant	4 minutes	5 minutes
Instant	20 minutes	5 minutes
Instant	25 minutes	5 minutes
Instant	20 minutes	5 minutes
	AVERAGE.	
----------	----------	4.1 minutes

An isotonic solution of sulfanilamide (0.9 per cent) was used on nasal polyp tissue. Six cases were studied. The cilia slowed after 20 to 112 minutes, and stopped in 36 to 119 minutes. The cilia recovered when washed with Locke's solution in every case. The average slowing time was 48 minutes, and average stopping time was 58 minutes.

TABLE 2. DATA ON SATURATED SOLUTION OF SULFANILAMIDE (0.9 PER CENT).

Cilia Slowed	Cilia Stopped	Cilia Recovered
20 minutes	46 minutes	Yes
112 minutes	119 minutes	Yes
40 minutes	44 minutes	Yes
47 minutes	55 minutes	Yes
35 minutes	48 minutes	Yes
34 minutes	36 minutes	Yes
	AVERAGE.	
48 minutes	58 minutes	

When freshly prepared 5 per cent isotonic solution of sodium sulfathiazole was used, the average slowing time was 31 minutes and average stopping time was 60.5 minutes. The cilia recovered, in half the cases, following immersion in Locke's solution. The P.H. of the 5 per cent solution of sodium sulfathiazole was 10.17, which may be a factor in the failure to recover in a number of instances.

TABLE 3. DATA ON 5 PER CENT SOLUTION OF SODIUM SULFATHIAZOLE (FRESHLY PREPARED) (ISOTONIC SOLUTION).

Cilia Slowed	Cilia Stopped	Cilia Recovered
25 minutes	130 minutes	No
50 minutes	80 minutes	No
20 minutes	50 minutes	Yes
35 minutes	126 minutes	Yes
15 minutes	25 minutes	Yes
15 minutes	25 minutes	Yes
30 minutes	35 minutes	Yes
55 minutes	65 minutes	No
53 minutes	70 minutes	No
40 minutes	50 minutes	No
20 minutes	35 minutes	Yes
23 minutes	35 minutes	No
	AVERAGE.	
31 minutes	60.5 minutes	

When 5 per cent sodium sulfathiazole (without sodium chloride to make it isotonic) was used, the average slowing time was 13.7 minutes, and the average stopping time 30 minutes. Comparison of the average time between action of the 5 per cent sodium sulfathiazole alone, with that of sodium sulfathiazole with sodium chloride to make it isotonic, would seem to indicate that the isotonic solution is less irritating.

TABLE 4. DATA ON 5 PER CENT SODIUM SULFATHIAZOLE WITHOUT SODIUM CHLORIDE TO MAKE IT ISOTONIC.

Cilia Slowed	Cilia Stopped	Cilia Recovered
15 minutes	36 minutes	Yes
15 minutes	24 minutes	Yes
10 minutes	33 minutes	Yes
15 minutes	27 minutes	No
	AVERAGE.	
13.7 minutes	30 minutes	

Use of a 2.5 per cent sulfadiazine in triethanolamine and butoben revealed that cilia slowed in four to nine minutes, and stopped at five to 10 minutes. All recovered when washed with Locke's solution. The average slowing time was 5.8 minutes and for stopping, 7.5 minutes.

TABLE 5. DATA ON 2.5 PER CENT SULFADIAZINE IN TRIETHYANOLAMINE AND BUTOBEN.

Cilia Slowed	Cilia Stopped	Cilia Recovered
5 minutes	7 minutes	Yes
9 minutes	10 minutes	Yes
7 minutes	8 minutes	Yes
5 minutes	7 minutes	Yes
5 minutes	8 minutes	Yes
4 minutes	5 minutes	Yes
	AVERAGE.	
5.8 minutes	7.5 minutes	

The cilia stopped in one to four minutes when triethanolamine and butoben (solvent only) were used. All six cases studied recovered when treated with Locke's solution. The average time for stopping was 2.1 minutes. The effect of the solvent was similar to that of the 2.5 per cent sulfadiazine in triethanolamine and butoben.

TABLE 6. DATA ON TRIETHANOLAMINE AND BUTOBEN (SOLVENT ONLY).

Cilia Stopped	Cilia Recovered
5 minutes	Yes
3 minutes	Yes
1½ minutes	Yes
1 minute	Yes
1½ minutes	Yes
1 minute	Yes
AVERAGE.	
2.1 minutes	

Sulfanilamide, sulfathiazole and sulfadiazine powders, left in contact for varying periods of time, caused no change in the ciliary action. Neither slowing nor stopping was observed. Application periods varied from five to 65 minutes.

TABLE 7. DATA ON SULFANILAMIDE POWDER.

Time Powder Applied	Result
5 minutes	No change
20 minutes	No change
20 minutes	No change
20 minutes	No change
20 minutes	No change
65 minutes	No change

TABLE 8. DATA ON SULFATHIAZOLE POWDER.

Time Powder Applied	Result
5 minutes	No change
20 minutes	No change
20 minutes	No change
20 minutes	No change
21 minutes	No change
33 minutes	No change
37 minutes	No change
55 minutes	No change

TABLE 9. DATA ON SULFADIAZINE POWDER.

Time Powder Applied	Result
11 minutes	No change
12 minutes	No change
20 minutes	No change
20 minutes	No change
22 minutes	No change
24 minutes	No change

The effects of two new vasoconstrictors also were studied and compared. These were 2-amino-heptane sulfate (Tuamine sulfate) in 0.5 per cent, 1 per cent, 2 per cent and 4 per cent solutions, and 4-methyl 2-amino-hexane sulfate (Fouramine sulfate) in 0.25 per cent, 1 per cent, 2 per cent and 4 per cent solutions.

The cilia recovered in all cases when 0.5 per cent 2-amino-heptane sulfate (Tuamine sulfate) was used. The cilia were slowed from 43 to 92 minutes and stopped from 54 to 115 minutes.

TABLE 10. DATA ON 0.5 PER CENT 2-AMINO-HEPTANE SULFATE (TUAMINE SULFATE).

Cilia Slowed	Cilia Stopped	Cilia Recovered
85 minutes	115 minutes	Yes
88 minutes	108 minutes	Yes
92 minutes	109 minutes	Yes
60 minutes	70 minutes	Yes
53 minutes	63 minutes	Yes
43 minutes	54 minutes	Yes
	AVERAGE.	
70.1 minutes	86.5 minutes	

Table 11 reveals that a 1 per cent isotonic solution of 2-amino-heptane sulfate (Tuamine sulfate) slowed the cilia in 30 to 57 minutes, and stopped their action in 75 to 170 minutes. Washing in Locke's solution brought recovery in only two of six cases. Average slowing time was 41.6 minutes, but average stopping time, 119 minutes, probably explaining the failure to recover.

TABLE 11. DATA ON 1 PER CENT ISOTONIC SOLUTION OF 2-AMINO-HEPTANE SULFATE (TUAMINE SULFATE).

Cilia Slowed	Cilia Stopped	Cilia Recovered
37 minutes	160 minutes	No
45 minutes	75 minutes	No
50 minutes	100 minutes	No
30 minutes	114 minutes	Yes
57 minutes	170 minutes	Yes
31 minutes	95 minutes	No
	AVERAGE.	
41.6 minutes	119 minutes	

Cilia recovered in four out of six cases, when 2 per cent solution of 2-amino-heptane sulfate (Tuamine sulfate) was used. The cilia were slowed by the solution in 17 to 34 min-

utes, and were stopped in 32 to 72 minutes. Average time for slowing was 23.1 minutes, and for stopping, 54.5 minutes.

TABLE 12. DATA ON 2 PER CENT ISOTONIC SOLUTION OF 2-AMINO-HEPTANE SULFATE (TUAMINE SULFATE).

Cilia Slowed	Cilia Stopped	Cilia Recovered
34 minutes	38 minutes	No
18 minutes	32 minutes	Yes
25 minutes	60 minutes	Yes
17 minutes	65 minutes	No
25 minutes	72 minutes	Yes
20 minutes	60 minutes	Yes
	AVERAGE.	
23.1 minutes	54.5 minutes	

There were no recoveries of ciliary action after treatment with Locke's solution when 4 per cent isotonic solution of 2-amino-heptane sulfate (Tuamine sulfate) was applied to the mucosa. In seven cases studied, observers noted that cilia slowed in 13 to 18 minutes and stopped in 28 to 40 minutes. The slowing time was observed after an average of 15.2 minutes, and the average stopping time was 33.4 minutes.

TABLE 13. DATA ON 4 PER CENT ISOTONIC SOLUTION OF 2-AMINO-HEPTANE SULFATE (TUAMINE SULFATE).

Cilia Slowed	Cilia Stopped	Cilia Recovered
15 minutes	36 minutes	No
15 minutes	40 minutes	No
16 minutes	31 minutes	No
16 minutes	28 minutes	No
18 minutes	33 minutes	No
14 minutes	35 minutes	No
13 minutes	31 minutes	No
	AVERAGE.	
15.2 minutes	33.4 minutes	

The cilia recovered in all cases when a solution of 0.25 per cent 2-amino-4-methylhexane sulfate (Fouramine sulfate) was used. The cilia were slowed from 60 to 175 minutes and stopped from 95 to 268 minutes.

TABLE 14. DATA ON 0.25 PER CENT 2-AMINO-4-METHYLHEXANE SULFATE (FOURAMINE SULFATE).

Cilia Slowed	Cilia Stopped	Cilia Recovered
93 minutes	108 minutes	Yes
60 minutes	95 minutes	Yes
63 minutes	128 minutes	Yes
163 minutes	193 minutes	Yes
98 minutes	268 minutes	Yes
175 minutes	255 minutes	Yes
	AVERAGE.	
108.6 minutes	174.5 minutes	

A 1 per cent solution of 2-amino-4-methylhexane sulfate (Fouramine sulfate) slowed the cilia in from 72 to 110 minutes and stopped action in 110 to 210 minutes. Activity slowed after an average of 97 minutes, and stopped on an average of 157 minutes. There were no recoveries in the six cases studied.

TABLE 15. DATA ON 1 PER CENT ISOTONIC SOLUTION OF 2-AMINO-4-METHYLHEXANE SULFATE (FOURAMINE).

Cilia Slowed	Cilia Stopped	Cilia Recovered
72 minutes	210 minutes	No
.....	120 minutes	No
.....	128 minutes	No
110 minutes	185 minutes	No
.....	110 minutes	No
110 minutes	190 minutes	No
	AVERAGE.	
97 minutes	157 minutes	

A 2 per cent isotonic solution of 2-amino-4-methylhexane sulfate (Fouramine) slowed the cilia after an average of 74 minutes and stopped action after an average of 207 minutes. Slowing occurred after periods of 61 to 90 minutes, and stopping after 165 to 255 minutes. There were no recoveries when the cilia were washed with Locke's solution.

TABLE 16. DATA ON ISOTONIC SOLUTION OF 2-AMINO-4-METHYLHEXANE SULFATE (FOURAMINE).

Cilia Slowed	Cilia Stopped	Cilia Recovered
85 minutes	255 minutes	No
65 minutes	190 minutes	No
75 minutes	255 minutes	No
61 minutes	171 minutes	No
90 minutes	210 minutes	No
70 minutes	165 minutes	No
	AVERAGE.	
74 minutes	207 minutes	

Average slowing time was 80 minutes, and average stopping time, 160 minutes when 4 per cent isotonic solution of 2-amino-4-methylhexane sulfate (Fouramine sulfate) was used. There was one instance of recovery, when the cilia were treated with Locke's solution. The cilia slowed in 50 to 110 minutes and stopped after 115 to 210 minutes.

TABLE 17. DATA ON 4 PER CENT ISOTONIC SOLUTION OF 2-AMINO-4-METHYLHEXANE SULFATE (FOURAMINE SULFATE).

Cilia Slowed	Cilia Stopped	Cilia Recovered
65 minutes	165 minutes	No
110 minutes	210 minutes	No
85 minutes	115 minutes	No
88 minutes	184 minutes	Yes
53 minutes	120 minutes	No
50 minutes	170 minutes	No
	AVERAGE.	
80 minutes	160 minutes	

In using a 2 per cent isotonic solution of ephedrine sulfate, all cilia recovered when washed with Locke's solution. The average slowing time was 85 minutes and the average stopping time was 138 minutes.

TABLE 18. 2 PER CENT ISOTONIC SOLUTION OF EPHEDRINE SULFATE.

Cilia Slowed	Cilia Stopped	Cilia Recovered
72 minutes	100 minutes	Yes
128 minutes	168 minutes	Yes
110 minutes	150 minutes	Yes
75 minutes	130 minutes	Yes
65 minutes	210 minutes	Yes
60 minutes	75 minutes	Yes
	AVERAGE.	
85 minutes	138 minutes	

The effects of three vasoconstrictors were compared. These were ephedrine, 2-amino-heptane sulfate (Tuamine sulfate) and 2-amino-4-methylhexane sulfate (Fouramine sulfate), each at a 2 per cent concentration in isotonic solution. The ephedrine slowed the cilia in 85 minutes, the 2-amino-heptane sulfate (Tuamine sulfate) in 23 minutes, and the 2-amino-4-methylhexane sulfate (Fouramine sulfate) in 74 minutes. In the same order, the cilia were stopped in 139, 54.5 and 204 minutes by the vasoconstrictors. Washing with Locke's solution restored activity of cilia affected by ephedrine and 2-amino-heptane sulfate (Tuamine sulfate), but there was no recovery of cilia stopped by 2-amino-4-methylhexane sulfate (Fouramine sulfate) in this concentration.

TABLE 19. COMPARISON OF THE THREE VASOCONSTRICTORS, EACH AT A 2 PER CENT CONCENTRATION IN ISOTONIC SOLUTION.

Vasoconstrictor	Cilia Slowed	Cilia Stopped	Cilia Recovered
2% Ephedrine	85 minutes	139 minutes	Yes
2% Tuamine sulfate	23 minutes	54.5 minutes	Yes
2% Fouramine sulfate	74 minutes	204 minutes	No

When a solution of penicillin, containing 5,000 units per cubic centimeter of normal saline was used, the average stopping time was 121 minutes. Recovery of the cilia occurred in all but two in the 22 cases studied.

TABLE 20. DATA ON PENICILLIN, 5,000 UNITS PER CUBIC CENTIMETER OF NORMAL SALINE.

Spec.	Time of Ciliary Movement Following Application of Penicillin	Recovery Following Application of Normal Saline
A	54 minutes	No
B	45 minutes	Yes
C	39 minutes	No
D	37 minutes	Yes
E	84 minutes	Yes
F	24 minutes	Yes
G	145 minutes	Yes
H	125 minutes	Yes
I	146 minutes	Yes
J	124 minutes	Yes
K	96 minutes	Yes
L	179 minutes	Yes
M	170 minutes	Yes
N	169 minutes	Yes
O	162 minutes	Yes
P	160 minutes	Yes
Q	162 minutes	Yes
R	109 minutes	Yes
S	158 minutes	Yes
T	159 minutes	Yes
U	158 minutes	Yes
V	172 minutes	Yes
	AVERAGE. 121 minutes	

COMMENT.

It is quite evident that these results are not absolutely conclusive, since the number of cases studied has been comparatively small.

The experiments do establish rather definitely that chlorbutanol, used as a preservative, is responsible for slowing and stopping ciliary action. They indicate that a freshly prepared isotonic solution of ephedrine sulfate, without chlorbutanol, is more satisfactory.

Sulfanilamide, sulfathiazole and sulfadiazine, in the powder form, had no effect on the cilia.

Saturated solutions of sulfanilamide in most instances had no permanent deleterious effect on the cilia.

The cilia did not recover after prolonged immersion in isotonic 5 per cent sodium sulfathiazole in about one-half of the cases. This may be due to the fact that the Solution had a PH of 10.17.

A 2.5 per cent solution of sulfadiazine in triethanolamine and butoben stops the cilia almost at once, but when washed ciliary activity is resumed. This effect is probably due to the solvent.

2-amino-heptane sulfate (Tuamine sulfate) in concentrations advised for clinical use (1 per cent) did not slow the cilia until an average of 41 minutes.

The average slowing time of the cilia was 97 minutes when 1 per cent 2-amino-4-methylhexane sulfate (Fouramine sulfate) was used.

Recovery of the cilia occurred in all instances when weaker solutions such as 0.5 per cent 2-amino-heptane sulfate (Tuamine sulfate) and 0.25 per cent 2-amino-4-methylhexane sulfate (Fouramine sulfate) were used.

It has been shown that penicillin is practically nonirritating when applied directly to the cilia of nasal mucosa. The solution used was 5,000 units per cubic centimeter of normal saline. This solution is much more concentrated than is ordinarily advocated for local application of the drug.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct an examination in Chicago at the Palmer House on Oct. 4-5-6-7, 1944.

FACIAL PARALYSIS ASSOCIATED WITH OTITIS MEDIA.*†

RUSSELL M. DECKER, M.D., Pasadena.

In order to understand the causes of facial paralysis associated with otitis media, it is best first to refresh our minds with the anatomy of the nerve and its relationship during its course through the temporal bone.

ANATOMY.

The nerve enters the internal auditory meatus lying above the cochlear and the vestibular divisions of the auditory nerve. At the fundus of the meatus, it turns and traverses the petrous bone in close relationship to the second spiral of the cochlear to enter the hiatus fallopii; here the geniculate ganglion is located. At this point the nerve trunk bends sharply backward and outward to enter the medial wall of the tympanic cavity. It then passes between the external semicircular canal above and the oval window below to the pyramid. At this point the nerve turns, passing vertically downward to emerge from the stylomastoid foramen.

CAUSES OF PARALYSIS.

Paralysis may result from lesions anywhere along the course of the facial nerve, whether centrally or peripherally located. If the lesion is of central origin, it will be situated in or near the facial nucleus. The various lesions which may cause central paralysis are swellings or tumor formations, hemorrhages, collections of pus, aneurysms, or granulomas and cicatricial contractions associated with tuberculosis, syphilis, etc. These central lesions frequently produce bilateral paralysis in contradistinction to the peripheral type, in which unilateral paralysis is the rule. If one side is involved in a central paralysis, the upper half of the face is not included because muscles which act bilaterally have representation on both sides

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†From the Department of Otolaryngology, University of Southern California.

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of the cortex. In the paralysis of central origin, other cranial nerves are usually also involved.

If the lesion is located within the temporal bone it is usually associated with infections of the middle ear or mastoid process.

Preoperative facial palsy occurs in association with the following: *1.* Acute suppurative otitis media, in which the inflammatory process extends through the tympanic wall of the fallopian canal. *2.* Acute suppurative mastoiditis, in which the nerve is directly exposed due to a dehiscence in the wall of the facial canal. *3.* Chronic suppurative otitis media, in which the necrosis involves the fallopian or the facial canal.

More rarely the palsy may be due to the following: *1.* Suppurative labyrinthitis, in which the necrosis extends through the horizontal semicircular canal to the facial canal. *2.* Otitis meningitis, in which the facial involvement comes from the associated suppurative labyrinthitis. *3.* Tuberculosis of the middle ear or mastoid process. *4.* Herpes zoster auricularis (Hunt syndrome).

Postoperative facial paralysis may be due to: *1.* Division or injury of the nerve during surgery. *2.* Several hours or days following surgery paralysis may occur due to trauma during the operation. *3.* If the tympanic wall of the facial canal is defective or unusually thin, paralysis may develop due to the pressure of gauze packing. *4.* In an infant, if the initial mastoid incision is too low, injury of the nerve may occur at its exit from the stylomastoid foramen.

OTHER CAUSES.

Tumors involving the nerve as it enters the internal auditory meatus usually involve the eighth nerve. Deafness and vertigo are associated with the facial paralysis in these cases.

In the internal ear the nerve is exposed to injury in cases of skull fracture.

The nerve is most exposed as it enters the fallopian canal and crosses the medial wall of the middle ear. In this region the canal wall is very thin as compared to the thick wall which protects it in the facial canal.

Pollak states that there are numerous vascular anastomoses between the nerve and the middle ear. In his opinion the

paralysis is not due to direct contagion but to irritative processes in the neurovascular apparatus. The vasomotor nerves to the vessels connecting the middle ear with the canal become the victims of toxic paralysis. This results in edema, possibly followed by degenerative changes in the nerve.

Kettel states that an important cause of facial palsy is a granulating inflammation in the neighborhood of the facial ridge.

An osteitis of the retrofacial cells progressing in spite of operation may bring on a palsy within a few hours or a few days.

LOCALIZATION OF THE SITE OF INJURY.

The site of the injury may be deduced by a study of the associated symptoms. A complete paralysis affecting all ramifications of the nerve is suggestive of severance of the nerve. Forehead movement on the affected side suggests a deeply seated lesion.

Partial paralysis occurs as a result of pressure on the nerve. This may be due to edema of the surrounding tissue, hemorrhage or serous exudate, gauze, a spicule of bone in the canal or granulations over the exposed nerve. Sometimes the nerve may be unequally affected, resulting usually in paralysis of the upper facial branches.

If injury of the nerve occurs above the chorda tympani, the sense of taste in the anterior two-thirds of the tongue on the same side is affected. If it occurs above the branch to the stapedius muscle, there results a severe tinnitus, hyperacusis and a confusion of sounds.

INCIDENCE.

The incidence of facial paralysis is rather low. Kettel reports 2 per cent in 13,175 cases of both acute and chronic otitis media. This is also the same per cent as observed by Persky and Pohlman.

In reviewing 400 cases of facial paralysis at the Los Angeles General Hospital during the past ten years, 67 per cent were due to general causes as cerebral hemorrhage, cerebral thrombosis-carcinoma of the parotid gland, skull fractures, plastic

operations about the ear, poliomyelitis, etc.; 18 per cent were true Bell's palsy; 15 per cent were associated with otitis media. Of this 15 per cent associated with otitis media, 8 per cent were in cases of acute otitis media; 6 per cent in preoperative cases; 2 per cent in postoperative cases; 7 per cent were in chronic otitis media; 4 per cent in preoperative cases; 3 per cent in postoperative cases.

After a study of these 400 cases, the following conclusions were drawn:

1. Facial paralysis developing in the first few days of an acute otitis media is probably from toxic causes, not an indication for mastoid operation unless accompanied by intracranial complications. *2.* If, however, it occurs late (after 10 days or so) in an acute otitis media, it is probably associated with bone necrosis and is an indication for surgical intervention. *3.* If a facial paralysis occurs with a chronic media, it is a definite indication for an immediate operation.

CONCLUSION.

1. Impairment of the facial nerve in its course through the temporal bone occurs at: *a.* fallopian canal, *b.* facial canal.

2. In the fallopian canal the wall is very thin. Edema of the nerve may subside after myringotomy and drainage is established.

3. In acute otitis media, paralysis coming on late is indicative of bone necrosis; subsequent surgery is indicated.

4. In chronic otitis media, a facial paralysis is a definite indication for surgical intervention.

65 North Madison Street.

AMERICAN BOARD OF OTOLARYNGOLOGY.

The next examination of the American Board of Otolaryngology will be held in New York City at the Waldorf-Astoria Hotel, June 1-2-3-4, 1944.

A TECHNIQUE OF SECONDARY SEPTAL RESECTION DEMONSTRATING REGENERATION OF THE SEPTAL CARTILAGE.*

LESTER W. EISENSTODT, M.D., Newark.

A great deal has been written about the classical submucous resection, but strangely enough the literature appears rather devoid of information on the subject of secondary operation which must be performed in many cases. The original operation should not be criticized, as conditions may have been present which prevented a thorough surgical performance. Certain errors, however, appear to be more common. Laszlo[1] strongly emphasizes, in his writings and teachings, that some surgeons fail to remove sufficient cartilage from the area near the dorsum so that the flaps cannot come together in a straight perpendicular line. Without this, an otherwise adequate operation may be rendered valueless.

TECHNIQUE.

At the New York Post-Graduate Medical School and Hospital, we use a 2 per cent cocaine and phenol pack for 10 minutes, followed by a 1 per cent novocaine solution containing eight drops of adrenalin per ounce. The novocaine is infiltrated into the whole of the septum. Infiltration causes additional anesthesia, hemostasis, and balloons out the mucosa. Dissection is thus made easier by thickening the flaps and diminishing the tendency toward button-holing. The columella and nasolabial angle are also injected for hemostasis and anesthesia since small branches of the infraorbital nerve supply the columella, and would not be blocked by the previous procedure alone. This measure also tends to alleviate the discomfort caused by pressure from the nasal speculum.

The location of the initial incision varies according to the nature of the deformity and the desire of the surgeon. If there is a high posterior deflection or an anterior perforation with a posterior deflection, one may employ a posterior inci-

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sion, making it anterior to the obstructing area. The incision should extend from as high up towards the dorsum as possible, down to the floor. On cutting through the mucosa one can feel the septal knife incise a substance which gives the impression of a cartilaginous consistency. This substance is the regenerated cartilage formed after the previous operation. Now, by means of a Freer elevator, the mucoperichondrial and mucoperiosteal flaps may be elevated. Although this incision may be satisfactorily employed, it is not advocated for most cases, due to the difficulty of keeping the flaps separated while operating.

In patients with large anterior spurs or lateral displacement of the anterior free end of the septum and in most cases of posterior deflection, I prefer to make the initial incision in the vestibular skin just midway between the anterior free end of the septum and the mucocutaneous junction. I find this approach is most suitable, as here there is a portion of the original cartilaginous septum which as a rule has not been manipulated in the previous operation. The incision extends from the highest point of the septum just anterior to the origin of the upper later cartilages, down to the nasal floor. It should be deep enough to go through the cartilage but should not penetrate the opposite vestibular skin. If this occurs, the process of elevating the mucoperichondrial flaps will be more difficult, as the anterior end of the septum will be free, more difficult to stabilize, and tend to buckle during blunt dissection; however, should one penetrate through to the opposite side, no perforation results, as this area can be easily sutured at the termination of the operation.

By means of a dull Freer elevator, the cleavage plane between the perichondrium and the cartilage is easily found, and this plane is followed back separating the original septal cartilage, regenerated septal cartilage, the perpendicular plate of the ethmoid, and the vomer from its mucoperichondrial and mucoperiosteal coverings. The mucoperichondrial and periosteal flaps are then elevated on the opposite side. The entire septum is now exposed as in doing a primary operation. Any obstruction caused by the regenerated cartilage, perpendicular plate, or vomer, may now easily be removed. This approach has the advantage of permitting resection of

most of the low anterior bony prominences on both sides which would be inaccessible by classical approaches.

The location of this initial incision is of utmost importance in following the cleavage planes of a regenerated septum which might be missed if a more posterior incision is employed (see Figs. 1 and 2).

If there is a spur which is anterior to the incision and is thus inaccessible, one can then make a small incision anterior to the spur and attempt to elevate the mucoperichondrium.

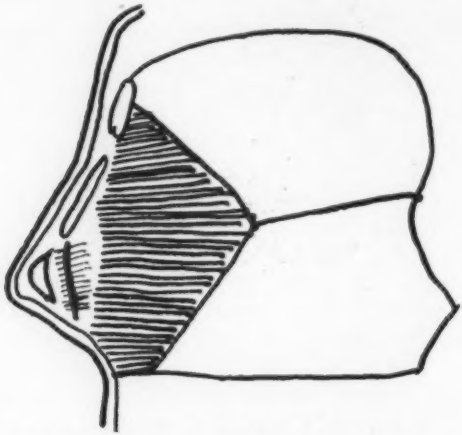

Fig. 1. Initial incision midway between anterior free end of septum and mucocutaneous junction.

If the membrane elevates intact, it is a simple procedure to knock off the spur with a chisel and remove it; however, if the flap tears, one simply mobilizes the spur by freeing its base. It can now be pushed to the midline, thus obliterating the obstruction.

Salinger[2] states that "the cartilage may be cut loose from its dorsal attachment, from its attachment to the vomer, and from the perpendicular plate posteriorly and safely replaced, even though its mucoperichondrium has been elevated on one side, as long as it still remains attached to soft tissue on the other side." The quadrangular cartilage may be almost completely excised and replaced in toto or in part without necro-

sis or other complication. Peer[3] also found that a unilateral attachment of the mucous membrane was unnecessary for the survival of the cartilage or the development of the nose.

REGENERATION OF THE NASAL SEPTUM.

To reiterate, if the septum is exposed in a secondary operation by the technique advocated, one may visualize an intact

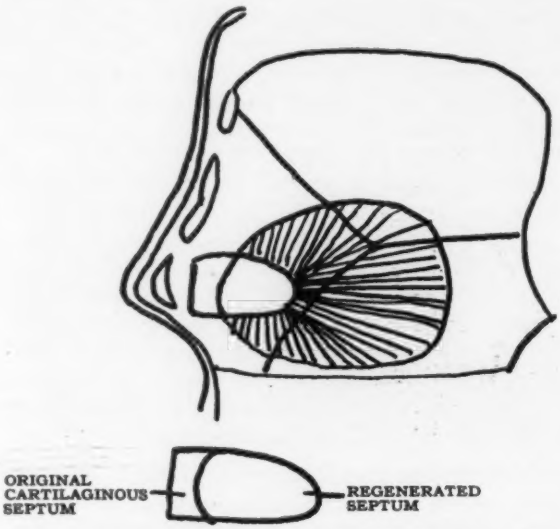

Fig. 2. Method of removing portion of original cartilaginous septum with attached regenerated septum.

cartilaginous and bony septum. This was observed in a series of nine secondary submucous resections. A search of the literature failed to reveal any confirmatory articles. This finding is corroborated by Dr. Alexander Zimany, Dr. Alexander Moshinsky and Dr. Alexander Laszlo, of the New York Post-Graduate Hospital.

In the performance of the typical submucous resection, the greater part of the quadrangular cartilage, with the exception of the dorsum and anterior free end, and portions of the perpendicular plate of the ethmoid and vomer, are excised. In performing the secondary operation, one recog-

nizes that all the component parts of the septum are present. The resected cartilaginous septum has been entirely replaced. This finding predicates intact bilateral mucoperichondrial flaps. If a flap is destroyed, it would be replaced by a fibrous tissue covering from the opposite intact flap. No regeneration is possible in an area where the mucoperichondrium has been destroyed in two places diagonally opposite each other and a perforation results. Maximow[4] states, "After a wound or excision of a portion of living hyaline cartilage in adult mammals, an independent regeneration of the cartilage does not take place according to most observers. This probably depends on the inability of the cells of mature cartilage to divide in the mammals. The defect is quickly filled by newly formed connective tissue which grows in from the perichondrium or nearest fascia. The fibroblasts of this granulation tissue become round and produce capsules around themselves and may become transformed into new cartilage cells. Accordingly in adult organisms, new cartilage tissue is formed by metaplasia of the loose connective tissues. Some cartilage cells may develop into osteocytes. . . . The fibroblasts of the connective tissue lose their spindle shape, changing into spherical cells, and thus are transformed directly into cartilage cells surrounded by capsules. The ability of the perichondrium to form cartilage persists, although latent in the adult organism."

My observation in these cases leads me to believe that each of the two mucoperichondrial flaps independently is capable of chondrogenesis and the formation of a separate septum providing the other flap is intact.

The newly formed nasal septum varies in individuals from a mere fibrous tissue formation to a complete cartilaginous or bony formation. Some specimens consisted of a whitish, flexible, hyalinized, fibrous tissue, others of a pinkish white, rather firm fibrous membrane with scattered islets of cartilage, while still others consist of thin plates of adult bone or cartilage. It is more difficult to strip the perichondrium from either side of the regenerated septal cartilage than from the original hyaline cartilage. These regenerated septa have exactly replaced the previously excised septa. It may also be noted that in this series of cases the perpendicular plate of the ethmoids and the vomers were intact, but, not having

performed the original submucous resections, I am not in a position to ascertain whether or not they had been previously excised; however, I believe that regeneration in the bony septum occurs as well as in the cartilaginous (see Fig. 3 microphotograph).

The process of septal regeneration occurs very rapidly and the fibrous tissue membrane is laid down before the fifth week.

The case of Mr. H. O., age 55, is well remembered. Five weeks previous to operation, he received a severe blow on the nose. He complained

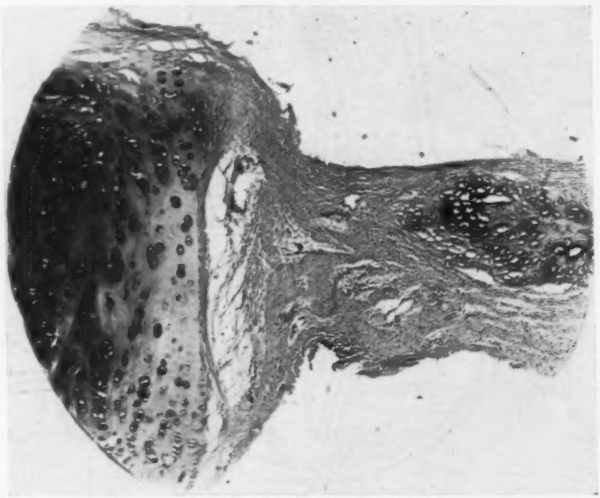

Fig. 3. Photomicrograph. Junction of original cartilaginous septum with fibrous regenerated septum containing a large island of cartilage.

of difficulty in breathing through his nose. Since the septum was very thick anteriorly, partially occluding both nostrils, the columella was separated from the anterior end of the septum and dissection was begun here. Three separate and distinct septa were found. The central one, badly deflected to both sides, was the original cartilaginous septum which was cloaked on either side by two newly formed membrano-cartilaginous flaps (see Fig. 4).

The discovery of two or three cartilaginous septa in the process of submucous resection is common, especially in post-traumatic deflections. Each new septum is derived from the perichondrium on that side, as each mucoperichondrial flap

in itself is capable of new septum formation. The thickness of the regenerated septum varies from 1 to 3 mm. and may serve as additional support for the bridge of the nose.

Rosenberger[5] describes the case of a double septum, one being osseous and blending with the perpendicular plate of the ethmoid; the other, cartilaginous, lying "against the bone like a slab of cheese in a sandwich." This, I believe, is a case of ossification of the original septal cartilage with a chondro-

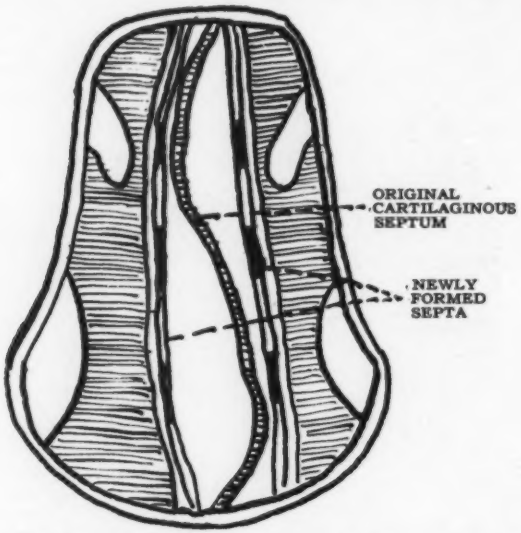

Fig. 4. Three distinct septa. The central one cartilaginous, the two lateral ones mebranocartilaginous.

genic proliferation from one of the mucoperichondrial flaps. I have had a similar case with complete ossification of the entire septum.

I believe that more children with severely deflected and fractured septa should be operated upon than is the present custom, both from a functional and cosmetic point of view. Most otolaryngologists defer septal operations until the age of 16. Peer[8] has found, after doing septal operations on children over the age of four, that the "nose appears to develop

quite normally as the child grows, and there may be some new formation of cartilage from the perichondrium." It has been the practice of Dr. Raymond Creasy at the New York Post-Graduate Hospital to perform conservative submucous resections on children for the past 10 years, and he has never found the operation to interfere in any way with the normal growth of the nose. I feel justified in stating that the chondrogenic power of the perichondrium is even greater in the young, and that the septal regeneration progresses to a much greater degree than in the adult. All the secondary operations performed were on adults between the ages of 20 to 55.

CONCLUSIONS.

1. A secondary submucous resection demonstrating the regenerated septal cartilage may successfully be performed in a manner similar to a primary operation.

2. The hyaline nasal septal cartilage is replaced after excision by a septum which may be membranous, membranocartilaginous, cartilaginous or bony in nature.

3. Submucous resections of the conservative type should be performed in children more often than is the present custom.

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31 Lincoln Park.

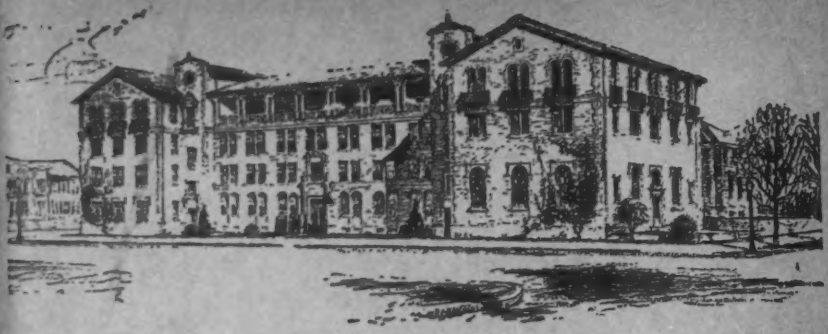
SUMMER COURSE IN AUDIOMETRY AND THE FITTING OF HEARING AIDS.

STATE UNIVERSITY OF IOWA.

The State University of Iowa will feature in its 1944 Summer Session, in addition to the regular program of graduate study leading to the M.A. and Ph.D. degrees in speech pathology or hearing conservation, an intensive four-weeks course in Audiometry and the Fitting of Hearing Aids, from June 26 to July 22. This will be a practical course for physicians, physicians' assistants, public health nurses, rehabilitation aides, speech correctionists and hearing conservation specialists in both civilian and military programs. There will be three hours daily of practical laboratory work in the Otologic Clinic of the University Hospital, supplemented by three hours of lectures on the various aspects of hearing conservation, including lip reading and speech training. The course will be under the direction of Dean M. Lierle, M.D., Head of the Department of Oral Surgery and Otolaryngology, and Chairman of the Committee on the Conservation of Hearing. He will be assisted by Scott Reger, Ph.D., and Loraine Anson, M.A. Supplementary instruction will be presented by the Speech Clinic staff, Wendell Johnson, Ph.D., Director; Charles R. Strother, Ph.D; Grant Fairbanks, Ph.D., and Jacqueline Keaster, M.A.

Running concurrently with this intensive course will be a Conference Series on Speech and Hearing Rehabilitation each week-end from June 23 to July 22. The Conferences will be conducted by the following visiting speakers: Bryng Bryngelson, Ph.D., University of Minnesota; Harold Westlake, Ph.D., Pennsylvania State Department of Education; Herbert Koepp-Baker, Ph.D., Pennsylvania State College; Raymond Carhart, Ph.D., Northwestern University; Walter Hughson, M.D., Abington, Pennsylvania Memorial Hospital. These speakers will discuss the various phases of speech and hearing rehabilitation in both its civilian and military aspects.

The Summer Speech Clinic for school children and adults will run from June 19 to July 28.



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